REMARKS

Claims 1-23, 30, 38 and 40-50 have been previously canceled without prejudice. Claims 27, 28, 51, 53, 54, 56, 57, 59 and 61 have been withdrawn from consideration by the Examiner under 37 CFR 1.142(b) as being drawn to non-elected inventions. Claims 24-29, 31-37, 39 and 51-61 are currently pending in this application.

Claims 55 and 58 have now been amended to include other inhibitory molecules. Support for this amendment is found on pages 40 and 43 of the specification.

The elected claims, as set forth above, are directed to methods for reducing hemostasis in a subject, and methods for the treatment or prevention of thrombotic disorders in subjects. All of the pending claims, listed above, are believed to read on these methods, and conform to the claims listed in the Office Action as reading on the elected invention.

The GenBank documents cited in a prior Information Disclosure Statement are enclosed with this Amendment. The requested information regarding the Genbank sequences is believed to be provided in the attached documents.

The specification has been amended to more closely conform the Title and Abstract with the claims currently pending in the application.

Claims 24-26, 29, 31, 32, 36, 37, 52, 55, 58 and 60 stand rejected under 35 U.S.C. § 102(e) as being anticipated by Wagner et al. (U.S. 2002/0040008 A1). This ground of rejection is respectfully traversed.

The Wagner et al. patent publication relates to the use of PSGL-1 to treat atherosclerosis. See Page 1, paragraph 4 of the Wagner et al. publication. As explained in the publication, atherosclerosis has nothing to do with blood coagulation, and it is not a thrombotic disorder. See pages 8 and 11 of the present specification which discusses procoagulant and thrombotic activity, respectfully, as those terms are used in the present invention.

In contrast, atheroscelrosis is the result of the accumulation of lipids and foam cells which penetrate arterial walls and, over extended periods of time, develop into lesions composed of muscle cells and lipid-filled macropghage and T cells. The lesions subsequently develop into fibrous plaques, or atherosclerosis. These plaques can rupture and subsequently result in a thrombosis. However, it is not the objective of the Wagner et al. publication to treat a thrombotic condition, but rather to treat atherosclerosis.

Atherosclerosis has nothing to do with coagulation as claimed in the present application. Applicants submit that one skilled in this art would not turn to the treatment of a long term condition, such as atherosclerosis, for guidance as to the treatment of blood coagulation disorders. In this regard, and in addition to the foregoing, applicants point out that several of the dependent claims are directed to specific thrombotic conditions, such as deep vein thrombosis (claim 33) and angina (claim 34), and that these specific conditions are not mentioned in the Wagner et al. publication.

Claims 24-26, 29, 31, 36-37, 52, 55, 58 and 60 stand rejected under 35 U.S.C. § 102(e) as being anticipated by Larsen et al. (U.S. Patent No. 5,840,679). This ground of rejection is traversed.

The Examiner states that Larsen et al. describes the use of PSGL for the treatment of myocardial infarction. However, the portion of the reference cited by the Examiner (col. 15, line 49 through col. 16, line 12) also discloses that {PSGL can be used to treat a variety of conditions, ranging from asthma to Grave's disease to multiple sclerosis. Furthermore, no basis or rational is provided for the use of PSGL to treat any of the conditions listed in this portion of the reference. For instance, what type of drug could be used successfully to treat all of the listed conditions, and how can one skilled in the art treat this disclosure with any degree of credibility? Absent such explanation, which is not provided, this disclosure is not entitled to any weight or credibility, and cannot thereby be considered to be enabling.

Claims 24-26, 29, 31-32, 35-37, 39, 52, 55, 58 and 60 stand rejected under 35 U.S.C. §102(e) as being anticipated by Nagy et al. (U.S. Patent No. 5,985,852). This ground of rejection is respectfully traversed.

The Nagy et al. reference is directed to the use of lipid compositions to prevent the binding of P-selectin or L-selectin to their corresponding ligands. The lipids described in Nagy. et al. are formed into a cross-linked sheet with carbohydrate and anionic binding regions. Thus, the lipids of Nagy et al. are essentially barriers interposed between the selectin and ligand, and are therefore not inhibitors as that term is defined in the present specification. Note, also, claims 25 and 26 which disclose, respectively, that the P-selectin inhibitor of this invention must be capable of decreasing the level of P-selectin in plasma, or decreasing the proteolytic cleavage of P-selectin from the cell surface. These are not inherent properties of the inhibitor, but actually

define the type of inhibitor useful in the invention in functional terms, and provide a basis for the selection of appropriate inhibitors. See also claims 36 and 39.

Although the Examiner states that the Nagy et al. reference discloses the use of PSGL-1 as an inhibitor, Nagy et al. only describes PSGL-1 in the "Background" section of the patent. Nagy et al. actually use lipids as barrier materials, and do not teach the use of PSGL-1 as inhibitors. Accordingly, Nagy et al. does not anticipate, teach or suggest the claims of the present invention.

Claims 24-26, 29, 31-37, 39, 52, 55, 58 and 60 stand rejected under 35 U.S.C. §103(a) as being obvious over Larsen et al. and Nagy et al. This ground of rejection is also traversed.

The Nagy et al. reference, described above, does not render the presently claimed invention obvious since it does not teach or suggest the use of inhibitors of P-selectin activity as claimed herein. As noted, the Nagy et al. reference is directed to highly specific lipid barriers interposed between the selectin and the ligand, and these barriers cannot be characterized as inhibitors.

Similarly, the Larsen et al. reference, also described above, is not enabling with respect to thrombotic disorders since the reference contains a broad listing of potential disorders unrelated in terms of mechanism of action or treatment modality.

The Examiner has acknowledged that neither Larsen et al. nor Nagy et al. disclose the specific thromboic conditions recited in present claims 33 and34, namely deep vein thrombosis and angina, respectively. However, the Examiner further states that the references disclose the treatment of a wide variety of thrombotic conditions which appear to have the common therapeutic feature of targeting the role of platelets. Applicants respectfully point out that this connection has not been made in the references, and appears to be merely conjectural.

In view of the foregoing facts and reasons, the present application is now believed to overcome the remaining rejections, and to be in proper condition for allowance. Accordingly, reconsideration and withdrawal of the rejections, and favorable action on this application, is solicited. The Examiner is invited to contact the undersigned at the telephone number listed below to discuss any matter pertaining to this application.

Respectfully submitted,

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